J HK Coll Radiol 2002;5:29-34

CASE REPORT

Acute Dural Sinus Thrombosis — Computed Tomography and Magnetic Resonance Imaging Features

KY Lau,[1] M Au-Yeung,[2] TH Tsoi,[2] DJ Roebuck,[3] LTH Tan[1]

[1]Department of Radiology, [2]Department of Medicine, Pamela Youde Nethersole Eastern Hospital, Hong Kong, [3]Department of Radiology, Great Ormond Street Hospital, London, United Kingdom

ABSTRACT

Dural sinus thrombosis is a diagnosis that is not commonly thought of in the first instance by radiologists and clinicians, especially when the clinical presentation is not specific. We describe the clinical presentation of three patients with acute thrombosis of the superior sagittal sinus, the left transverse sinus, and the right cavernous sinus, respectively. Compared with computed tomography, magnetic resonance imaging is more diagnostic of this condition. Magnetic resonance venography may not be necessary for the diagnosis of acute dural sinus thrombosis, but is helpful in cases of cortical or deep vein thrombosis and chronic thrombosis.

Key Words: *Cerebrovascular accident, Computed tomography, MRI, Thrombosis*

INTRODUCTION

Dural sinus thrombosis (DST) is a rare cause of stroke. The diagnosis can be difficult, because of its non-specific clinical manifestations and radiological findings. Delay in diagnosis may lead to venous congestion, venous infarction, and even death. Early and accurate diagnosis of this condition is therefore important. The clinical presentation of three patients with DST will be described and the CT and MRI findings will be discussed.

CASE REPORTS

Case 1

A 43-year-old Chinese woman presented with a 2-day history of left upper limb numbness, and a 1-day history of severe occipital headache. On the day of admission, she had sudden onset of left upper limb weakness, but sparing the left hand. This was followed by loss of consciousness for 5 minutes, retrograde amnesia, and drowsiness. There was no convulsion, joint pain, history of trauma, or hypertension. She had been on oral contraception for 7 years for acne and gynaecological problems. She was a non-smoker and was not diabetic. Physical examination revealed Grade I weakness of the left upper limb. The right upper limb and both lower limbs showed normal power. Fundoscopic examination showed no papilloedema.

Non-contrast CT (NCCT) of the brain on the second day of admission showed a subtle, poorly-defined, hypodense area in the right frontal lobe with effacement of the adjacent cerebral sulci, and without intracranial haemorrhage or midline shift (Figure 1). MRI performed on the fourth day of admission demonstrated a poorly-defined area in the right superior frontal gyrus and the right precentral gyrus with hypointense signal on T1-weighted images, hyperintense signal on T2-weighted images, mild gadolinium enhancement, and effacement of the adjacent cerebral sulci. Loss of the normal flow void in both T1- and T2-weighted images and small intraluminal filling defect in the post-gadolinium images in the anterior part of the superior sagittal sinus were considered diagnostic of thrombosis (Figure 2). Follow up NCCT on the day after MRI did not provide additional information. Her blood coagulation parameters were normal.

She was treated with low-molecular-weight heparin (LMWH) for 2 weeks, followed by warfarin. She made a good recovery, with only mild weakness of the left upper limb at 6 months.

Correspondence: *Dr. KY Lau, Department of Radiology, Pamela Youde Nethersole Eastern Hospital, 3 Lok Man Road, Chai Wan, Hong Kong.*
Tel: (852) 2515 3194; Fax: (852) 2595 6169;
E-mail: drkylau@yahoo.com

Submitted: 8 October 2001; Accepted: 30 November 2001.

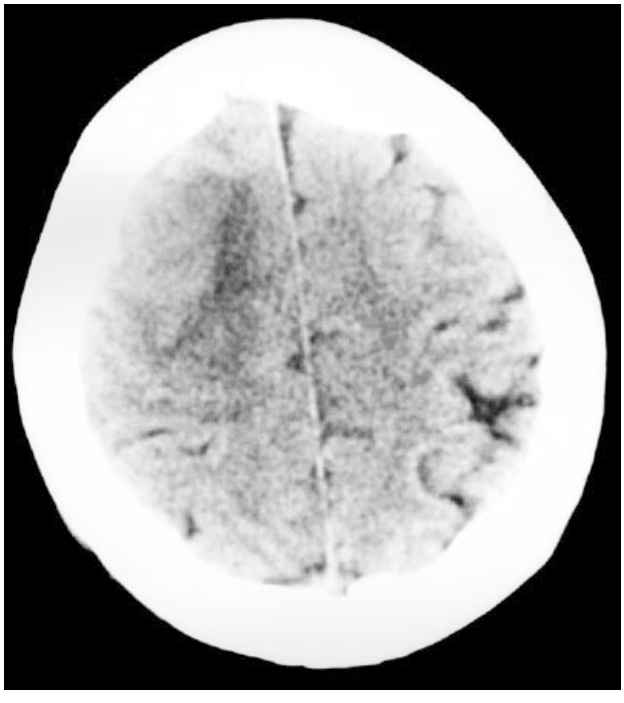
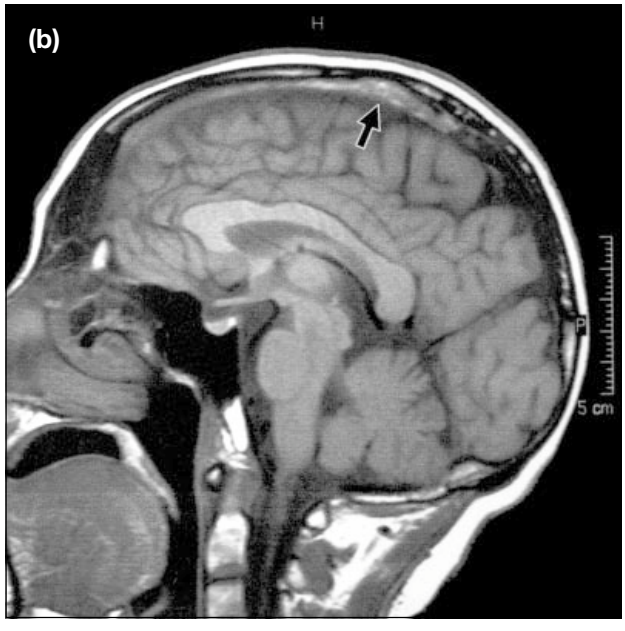
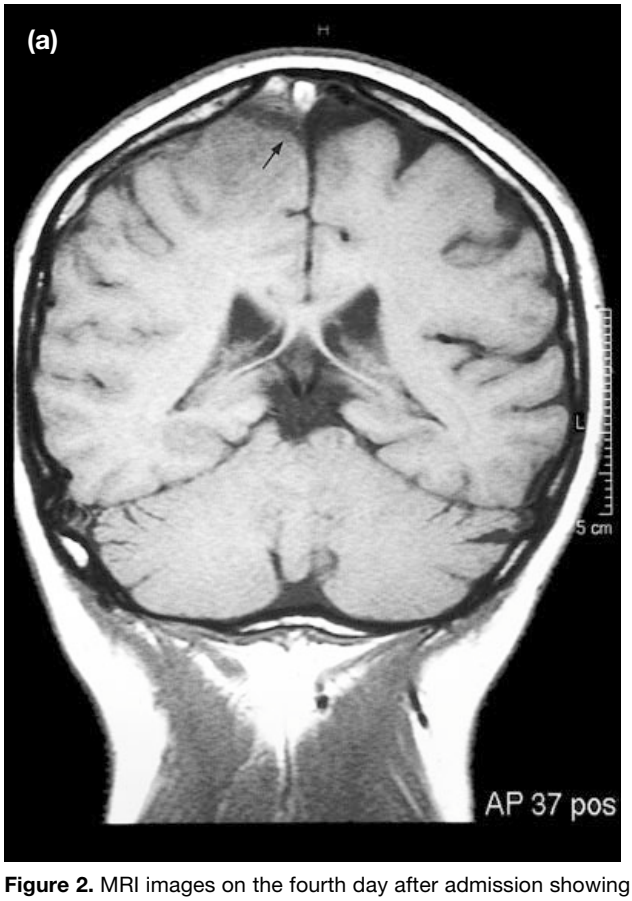


Figure 1. Axial CT scan revealing a subtle, poorly-defined, non-specific, hypodense area in the right high frontal lobe with effacement of the adjacent cerebral sulci.

Case 2

A 39-year-old Chinese woman presented with headache, vomiting, and drowsiness. In the emergency room, she had a first episode of generalised tonic-clonic





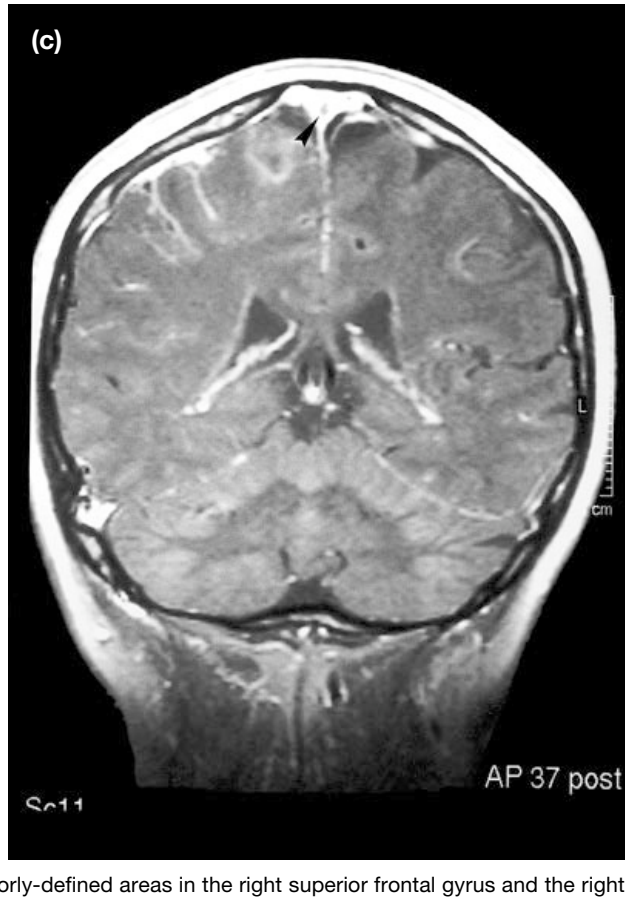


Figure 2. MRI images on the fourth day after admission showing poorly-defined areas in the right superior frontal gyrus and the right precentral gyrus. (a) Coronal T1-weighted image; (b) sagittal T1-weighted image. The lesion is parasagittal in location with effacement of the adjacent cerebral sulci. Loss of the normal flow void (arrow) is noted in the superior sagittal sinus; (c) post-gadolinium coronal T1-weighted image. A small intraluminal filling defect (arrowhead) compatible with thrombus is noted in the superior sagittal sinus.

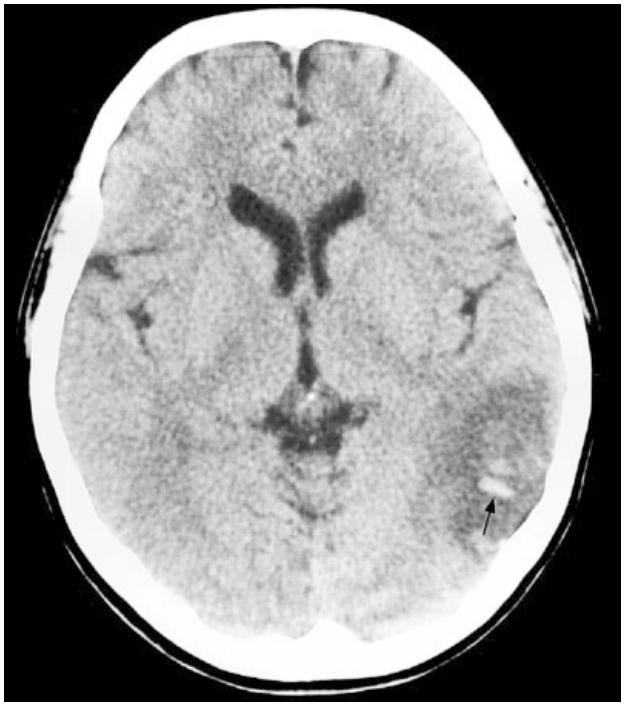


Figure 3. Follow-up CT scan 2 days later. There was a further increase in the size of the poorly-defined, hypodense area in the left temporo-occipital region with subcortical haemorrhagic foci (arrow).

convulsion, which was controlled by intravenous diazepam. There was no fever or focal neurological deficit. Fundoscopic examination revealed no retinal haemorrhage or papilloedema. She had been on oral contraception for 10 years, and (unknown) herbal medicines for weight reduction for 2 years.

Emergency NCCT of the brain showed a poorly defined hypodense area in the left temporo-occipital region with effacement of the adjacent cerebral sulci. The diagnosis was initially thought to be either encephalitis or subacute infarct. Follow-up NCCT of the brain 2 days later showed a further increase in the size of the hypodense area, with subcortical haemorrhagic foci (Figure 3). MRI of the brain was performed 3 days after presentation. The left temporo-occipital lesion was hypointense on T1-weighted images and hyperintense on T2-weighted images, with effacement of the adjacent sulci, mass effect, small foci typical of blood degradation products, and loss of the normal flow void in the left transverse sinus (Figure 4).

She was treated first with LMWH and subsequently with warfarin. She made an uncomplicated recovery, without further seizure. MRI of the brain performed at 3 months showed considerable resolution of the oedema, with mild focal gyriform increase in the signal intensity in the left temporo-occipital region. There was persistent loss of the flow void in the left transverse sinus.

Case 3

A 71-year-old Chinese woman presented with nausea and vomiting on the day of admission. She described a dull, aching, non-radiating, central, abdominal pain. There was no fever, chills, rigor, or diarrhoea. She had been treated for diabetes mellitus with oral hypoglycaemic drugs for 10 years. Physical examination

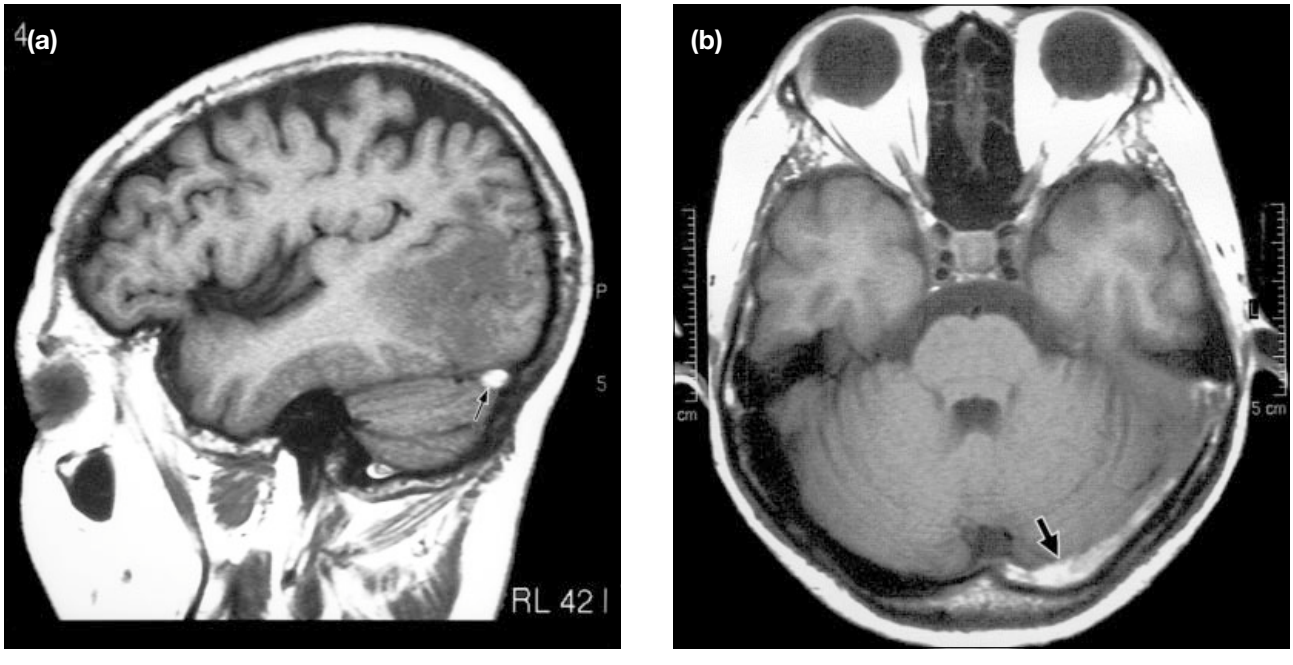


Figure 4. Three days after presentation. Sagittal T1-weighted image (a) and axial T1-weighted image (b) showing loss of the normal flow void in the left transverse sinus (arrow) and effacement of the adjacent sulci.

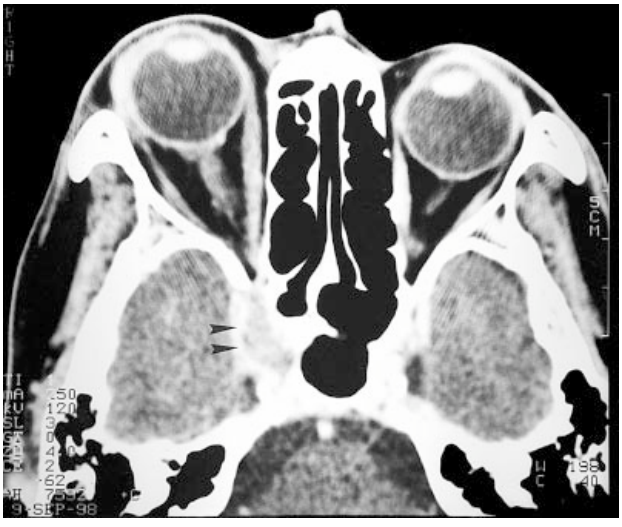


Figure 5. Axial CT scan of the orbits with contrast showing mild right proptosis and a filling defect in the prominent right cavernous sinus, consistent with thrombus (arrowheads).

revealed mildly decreased skin turgor and dry oral mucosa. Her arterial blood pressure was normal. She developed a right-sided abducens nerve palsy and headache on the second day of admission.

NCCT of the brain showed moderate swelling of the right cavernous sinus. On the following day, she developed a red right eye with mild exophthalmos and clinically obvious palsies of the right oculomotor, trochlear, ophthalmic, and abducens nerves. Contrast-enhanced CT (CECT) of the orbits on the next day showed an intraluminal filling defect in the right cavernous sinus, consistent with thrombosis (Figure 5). This diagnosis was supported by MRI, which was performed on the third day after presentation, and showed marked prominence of the right cavernous sinus with slight hypointensity on T1-weighted images and slight hypointensity on T2-weighted images. An intraluminal filling defect was present on the CECT images (Figure 6). She was treated with LMWH, penicillin G, cloxacillin, and a third generation cephalosporin. There was marked decrease in proptosis and chemosis in the following week. She was discharged with partial right oculomotor and abducens nerve palsies. At 4 months, there was no clinically detectable proptosis or diplopia.

DISCUSSION

DST may be associated with one or more identifiable predisposing factors (Table 1).[1] In the series published by Tsai et al, however, no cause was identified in 13 of 29 patients.[2] Two of our cases were associated with oral contraceptive use. The third patient was diabetic and presented with clinical signs of dehydration. However, the presenting signs and symptoms may not be specific,

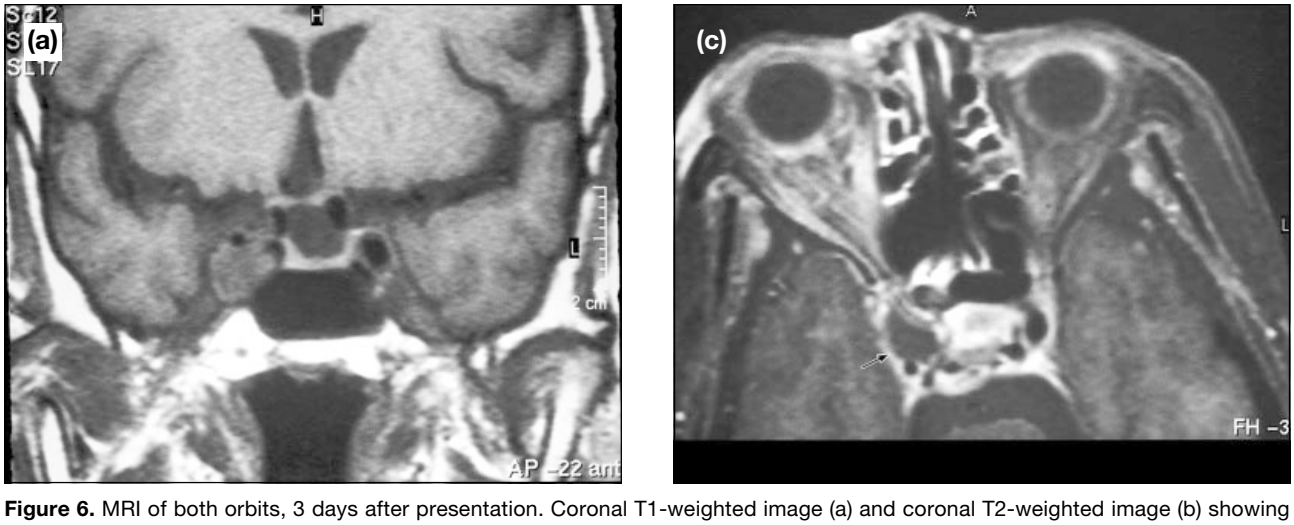


Figure 6. MRI of both orbits, 3 days after presentation. Coronal T1-weighted image (a) and coronal T2-weighted image (b) showing hypointense signal in the prominent right cavernous sinus; (c) post-gadolinium axial T1-weighted image showing the filling defect in the right cavernous sinus (arrow). Special note is made of the absence of parenchymal abnormality.

Table 1. Factors predisposing to cerebral venous thrombosis.[1]

Drugs	Oral contraceptives, androgens, L-asparaginase, ε-aminocaproic acid
Medical conditions and pregnancy	
Pregnancy	Pregnancy and puerperium
Hypercoagulable states	Protein S deficiency, protein C deficiency, antiphospholipid antibodies
Haematologic disorders	Polycythemia, sickle cell anaemia
Autoimmune disease	Sjogren syndrome, Behçet syndrome
Other medical conditions	Severe dehydration, homocystinuria, nephrotic syndrome
Infection	
Local	Otitis media, suppurative sinusitis
Systemic	Acquired immunodeficiency syndrome (AIDS), disseminated tuberculosis
Trauma	Head injury
Iatrogenic	Internal jugular vein cannulation, post-operative
Neoplasms	Central nervous system tumour, non-central nervous system tumour

and this may lead to a delay in the diagnosis. The most common symptoms include headache, lethargy, decreased conscious level, disturbance of vision, convulsion, and focal neurological deficit.[2] A slight female predominance has been observed in adult patients,[2,3] possibly related to certain risk factors, such as pregnancy[4,5] and oral contraceptive use.[6,7] DST may also affect infants[2,8] and children.[9,10] Various CT signs of cerebral venous thrombosis (CVT) have been described.[11-14] Direct signs include the *cord sign*, an area of hyperdensity on NCCT that represents the thrombosed cortical vein, and the *empty delta* sign, a filling defect in the sinus on CECT. Indirect signs include intense enhancement of dural structures, and non-haemorrhagic or haemorrhagic venous infarcts. Subcortical haemorrhage (seen as a hyperdense focus in the subcortical white matter) may be the only abnormality on NCCT in acute DST.[4] CT findings may, however, be normal.[10-14] The initial NCCT findings in all our patients were non-specific. The presence of subcortical haemorrhage in the follow-up NCCT of Case 2 should have raised the suspicion of DST. In Case 1, follow-up NCCT revealed no additional information. In Case 3, CECT showed diagnostic findings of cavernous sinus thrombosis.

The most reliable finding of DST on MRI is loss of the flow void in the affected sinus. According to Isensee et al, 4 stages of evolution of thrombosis may be observed.[15] Acute thrombosis (days 1 to 5) is isointense on T1-weighted images and hypointense on T2-weighted images. In the subacute stage (up to day 15), the thrombus is strongly hyperintense on both T1- and T2-weighted images, because of the presence of methaemoglobin. In the third week after the onset of symptoms, the thrombus shows decreased signal in all sequences, with increasing inhomogeneity due to recanalisation. The late stage is characterised by either the reconstitution of blood flow or the persistence of a residual thrombus. Yuh et al observed 3 major patterns of brain abnormality at MRI in patients with DST: mass effect without associated abnormal signal on T2-weighted images; mass effect with associated abnormal signal on T2-weighted images (and/or potentially reversible ventricular dilatation); and an intraparenchymal haematoma with surrounding oedema.[3]

In occlusive disease of the venous sinuses, breakdown of the blood-brain barrier (as shown by vasogenic oedema and abnormal parenchymal enhancement) does not always occur, and brain swelling can persist for up to 2 years, with or without abnormal signals on T2-weighted images.[3] Abnormal signals on T2-weighted images, possibly due to interstitial oedema (most likely related to raised intraventricular pressure), may be reversible, and does not always indicate infarction, as in Case 2.[3] Tsai et al have devised a grading system to assess the severity of acute DST at MRI (Table 2),[2] and this appears to have prognostic significance. In their study, parenchymal changes were reversible up to Stage III if thrombolytic treatment was used. Beyond stage III, there were some residual changes, even after thrombolysis. All of the Stage V patients died. Tsai et al therefore recommended that patients with Stage I DST should only be treated with anticoagulants, but followed by prompt thrombolysis if there is clinical deterioration. All other stages should be treated with thrombolysis.[2] According

Table 2. MRI grading of acute dural sinus thrombosis (after[2]).

Stage	Characteristic
I	No parenchymal changes
II	Sulcal effacement and mass effect, without signal change to indicate oedema
III	Attenuation and signal intensity changes compatible with mild to moderate oedema
IV	Severe oedema, with or without small haemorrhage
V	Massive oedema and/or haemorrhage

to Tsai's classification, our Cases 1 and 2 were Stage III and IV DST, respectively. Both patients recovered, however, after treatment with oral anti-coagulation alone. Prompt anticoagulant therapy, and possibly the degree of collateralisation, may be critical factors.

Venous infarctions are characteristically subcortical in location and occur at sites that do not correspond to any major arterial distribution. The sites of parenchymal changes usually reflect thrombosis in the adjacent dural sinus, as in our first 2 patients. This is not always the case, however. Tsai et al reported 2 patients with bilateral basal ganglia oedema, one of whom had superior sagittal sinus thrombosis, and the other thrombosis of the deep cerebral veins.[2] DST in the posterior fossa may cause supratentorial changes in addition to oedema or haemorrhage of the cerebellum and brain stem.[2] The transverse sinus (alone or in combination with other dural venous sinuses) is most commonly involved, followed by the superior sagittal sinus.[2,15] Even when thrombosis occurs in the midline superior sagittal sinus, subcortical haemorrhage is often unilateral.[15] In Case 1, who had thrombosis of the superior sagittal sinus, the parenchymal abnormality occurred in the right frontal lobe. This phenomenon may be related to asymmetrical distribution of collaterals on each side of the thrombosed sinus. However, no parenchymal abnormality was detected by CT or MRI in our patient with thrombosis of the right cavernous sinus. This may be due to the presence of a rich venous network around the cavernous sinuses.

It has been suggested that the use of iodinated contrast media for angiography and CECT is potentially dangerous in patients with DST,[17] because it may lead to dehydration and aggravation of a hypercoagulable state. Angiography has the additional disadvantage of being an invasive procedure. The superiority of MRI over CT in the detection of DST is well-established.[17,18] MRI is highly sensitive to blood flow, and can accurately demonstrate any associated parenchymal abnormality. Magnetic resonance venography (MRV) has also been found to be helpful.[5,16] According to Isensee et al, older thrombus or occlusion of cortical veins is best diagnosed by angiography.[16] However, all of our patients presented acutely, and there were no difficulties in making the diagnosis of DST by MRI, even without supplementary MRV.

In conclusion, DST should be suspected in patients with known risk factors who present with neurologic symptoms. The presence of subcortical haemorrhage or focal cerebral oedema at CT or MRI in patients with a non-specific clinical presentation should alert the radiologist to the possibility of DST. MRI should be performed promptly if CT has been the initial investigation and is not diagnostic. MRV is helpful, but may not be needed to reach the diagnosis of DST in acute cases.

ACKNOWLEDGMENTS

We thank Shalla Liu and Karman Wong for their secretarial support.

REFERENCES

1. Braunwald E, Fauci AS, Kasper DL, Hauser SL, Longo DL, Jameson JL. Harrison's principles of internal medicine (15th edition). New York: McGraw-Hill, 2001.
2. Tsai FY, Wang AM, Matovich VB, et al. MR staging of acute dural sinus thrombosis: correlation with venous pressure measurements and implications for treatment and prognosis. Am J Neuroradiol 1995;16:1021-1029.
3. Yuh WT, Simonson TM, Wang AM, et al. Venous sinus occlusive disease: MR findings. Am J Neuroradiol 1994;15:309-316.
4. Carroll JD, Leak D, Lee HA. Cerebral thrombophlebitis in pregnancy and the puerperium. Q J Med 1966;35:347-368.
5. Budak F, Bolukbasi O, Demirci A, Ozmenoglu M, Komsuoglu B. Cerebral dural sinus thrombosis. A case report. Angiology 1995;46:541-545.
6. Scarpa M, Colombo A, Sorgato P, Panzetti P, Crisi G, Fabbri G. Magnetic resonance imaging in cerebral sinus thrombosis. Report of a case. Ital J Neurol Sci 1990;11:289-292.
7. de Bruijn SF, Stam J, Koopman MM, Vandenbroucke JP. Case-control study of risk of cerebral sinus thrombosis in oral contraceptive users and in carriers of hereditary prothrombotic conditions. The Cerebral Venous Sinus Thrombosis Study Group. BMJ 1998;316:589-592.
8. Konishi Y, Kuriyama M, Sudo M, Konishi K, Hayakawa K, Ishii Y. Superior sagittal sinus thrombosis in neonates. Pediatr Neurol 1987;3:222-225.
9. Schwab FJ, Peyster RG, Brill CB. CT of cerebral venous sinus thrombosis in a child with homocystinuria. Pediatr Radiol 1987; 17:244-245.
10. Purvin V, Dunn DW, Edwards M. MRI and cerebral venous thrombosis. Comput Radiol 1987;11:75-79.
11. Chiras J, Bousser MG, Meder JF, Koussa A, Bories J. CT in cerebral thrombophlebitis. Neuroradiology 1985;27:145-154.
12. Buonanno F, Moody D, Ball R, Lester D. Computed cranial tomographic findings in cerebral sino-venous occlusion. J Comput Assist Tomogr 1978;2:281-290.
13. Buonanno F, Moody D, Ball R. CT findings in cerebral sinovenous occlusion. Neurology 1982;12:288-292.
14. Kingsley D, Kendall B, Mosely I. Superior sagittal sinus thrombosis, an evaluation of changes demonstrated on computed tomography. J Neurol Neurosurg Psychiatry 1978;41:1065-1068.
15. Keiper MD, Ng SES, Atlas SW, Grossman RI. Subcortical haemorrhage: marker for radiographically occult cerebral venous thrombosis on CT. J of Comput Assist Tomogr 1995;19:527-531.
16. Isensee C, Reul J, Thron A. Magnetic resonance imaging of thrombosed dural sinuses. Stroke 1994;25:29-34.
17. Harris TM, Smith RR, Koch KJ. Gadolinium DTPA enhanced MR imaging of septic dural sinus thrombosis. J Comput Assist Tomogr 1989;13:682-684.
18. Padayachee TS, Bingham JB, Graves MJ, Colchester AC, Cox TC. Dural sinus thrombosis. Neuroradiology 1991;33:165-167.